XVI
VOL. ~~XIV~~. July, 1889. No. 7

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

A REPORT OF A CASE OF SYRINGO-MYELIA.[1]

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FOR the opportunity of presenting this case I am indebted to Professor Francis Delafield, who gave me the material, with the clinical history and his notes of the autopsy.

Clinical History.—Lawrence B——; farmer; æt. 47; parents died of consumption; had been in good health with the exception of one slight attack of acute rheumatism in the hands until twenty-one months before death. He then noticed a soreness in the epigastrium and a numbness in the left foot and leg. These symptoms grew worse, and the right leg and foot also became numb.

For nine months after the commencement of the symptoms, the man was able to work on a farm, but could not do heavy lifting, and the jar of riding in a wagon made him numb from his waist down. At the end of this time he began to have a girdle sensation, and the motor and sensory symptoms in the legs had increased until he was hardly able to walk alone. The sphincters were normal and there were no symptoms in the upper part of the body.

During the *twelfth month* of the illness the condition was as follows: Occasional temporary incontinence of

[1] One of the essays for which the Cartwright prize was awarded in 1889.

urine lasting a day or two. Sensation normal in the back, but diminished in both legs, especially the left. Patellar reflexes slightly exaggerated, plantar reflexes diminished. Diaphragmatic and cremasteric reflexes absent. Patient cannot walk alone and drags the left foot. Romberg's symptom present. Ankle clonus absent. No rigidity of the legs. There were also periods of temporary improvement during this month.

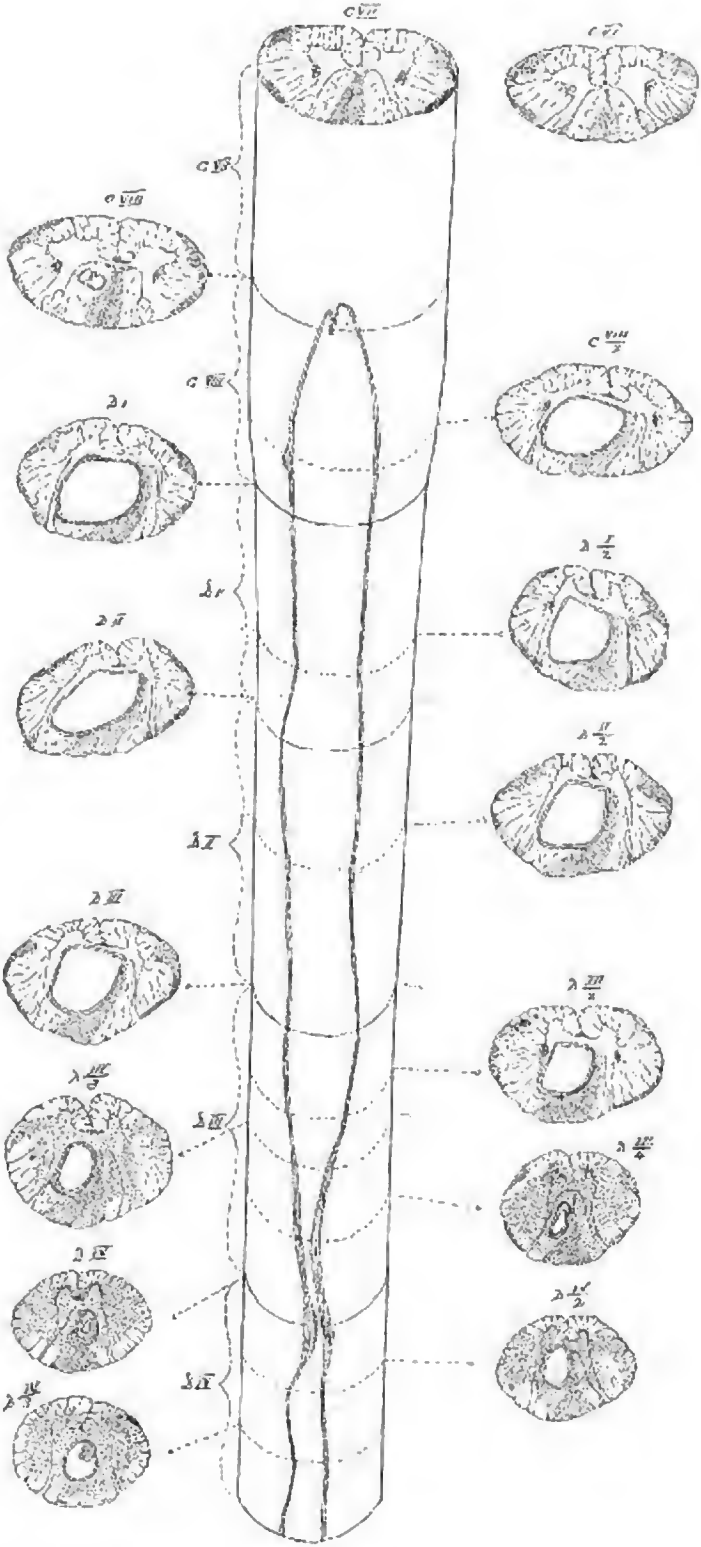
During the *thirteenth and fourteenth months* of the illness the following notes were recorded: Patient remains in bed most of the time. He can walk only with firm support; but on some days there is transient improvement, and he is then able to be up and move about. The sphincters are controlled, except occasionally. There is daily œdema of the feet, subsiding at night. The girdle sensation is diminished, and is at times absent. The patellar reflex is at times normal, at times exaggerated; the diaphragmatic reflex is sometimes present, sometimes absent; the plantar reflex is diminished, the cremasteric is absent and the ankle clonus is present.

From the *fifteenth to the eighteenth months* of the disease, there was very little change in the man's condition. The daily œdema of the feet, subsiding at night, continued. He could stand for a short time with his feet far apart. Constipation and slight dribbling of urine. The girdle sensation was constantly present. There was exaggeration of the patellar reflexes, and diminution or absence of the cremasteric reflexes. The nutrition was good. Sensation above the umbilicus was normal.

During the *nineteenth and twentieth months* there was in addition to the other symptoms considerable pain at times in the feet, and in the umbilical and hypogastric regions.

During the *last month* of the sickness bed sores developed, and the legs became very much swollen and œdematous. Four days before death the temperature rose to 103.5. The symptoms did not extend above the umbilicus.

Autopsy.—Body moderately emaciated. The thoracic and abdominal viscera were normal except the right lung, which was coated with fibrin, and the bronchi of its un-


C VIII
C VII
C VIII
C VIII/2
λ I
λ I/2
λ II
λ II/2
λ III
λ III/2
λ III/2
λ III/4
λ IV
λ IV/2
λ IV/2

aerated lower lobe contained pus. A fusiform enlargement of the spinal cord, caused by a reddish gray tumor filling up a large part of its transverse diameter as a solid plug, begins near the middle point of the dorsal region, and extends downward four cm. Extending about eight cm. above the tumor and about five cm. below it, is a centrally situated tubular cavity with a thin smooth translucent wall, varying from two mm. to three-quarters of a centimetre in diametre.

Projecting from the surface of the cord over the lower half of the tumor are about sixty small grayish white nodules, from one to one and a half mm. in diameter. Some of them, the smaller ones, lie scattered about on the posterior and lateral surfaces of the cord, and are fused with the root strands. A few of them spring from the cord at the exit of the anterior nerve roots. The majority of the nodules, including the larger ones, surround the filaments of the posterior roots at or near their entrance into the cord. Several of the nodules in the latter position extend for some distance (about two segments) below the tumor. The brain could not be examined. The cord was hardened in Müller's fluid, and afterwards in alcohol; it was then divided into segments corresponding to the insertions of the anterior nerve roots. The segments were carefully measured, and Fig. 1 was constructed from these measurements.

Microscopical Examination.—Above the tumor (Fig. 1), the posterior median, the direct cerebellar column, and the antero-lateral ascending columns of Gowers are degenerated; below the tumor there is a degeneration of the crossed pyramidal tracts. Above the upper third of the fourth dorsal segment, the inner four-fifths of the column of Goll are deflected to the right by the large cavity. The column of Gowers is larger on the right side in the sections D II-2 and D III (Fig. 1). In the other sections these columns are nearly symmetrical and equal.

The upper limit of the cavity is about two mm. above the superior surface of the eighth cervical segment (Fig. 1). The uppermost portion of the cavity is lined by a thin mem-

brane of neurogliar tissue, composed of larger and smaller branching neuroglia cells and small spheroidal and oval cells lying in a network of fibres which unites with the surrounding neuroglia with a more or less distinct line of demarcation (Fig. 2). In some places the elements of this membrane are packed closely together, so that in section it has a rather dense appearance; but in the greater portion of its extent the cells and fibres are loosely arranged, so that it has a porous meshwork structure as in Fig. 2. This

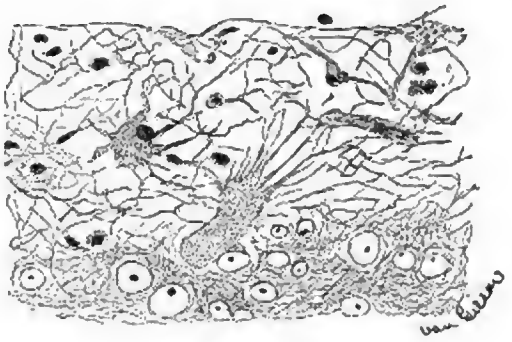


Fig. II.

thin membrane—which, for the sake of brevity, will be spoken of as the limiting membrane of the cavity—is the simplest form of the wall of the cavity, and is the only wall the cavity has in a considerable portion of its extent, viz., in the upper two-thirds of C VIII., in D I., D II., in the upper third of D III., and in the greater part of D IX. and D X.

This limiting membrane also lines the tubular cavity in the lower third of the tumor.

In other places the wall of the cavity is in a state of hyperplasia and is more complex in structure. In these places (indicated by the thickened vertical outline of the cavity in Fig. 1) the limiting membrane is sometimes present and sometimes altogether absent.

In the section C VIII. (Fig. 1) the cavity is bifurcated, appearing in the apex of the column of Burdach as two small circular spaces (1 mm. in diameter) surrounded by

the limiting membrane. The gray matter with its ganglion cells is normal, except that a large inner portion of the cervix and head of the posterior horns is replaced by a closely tangled, very fine filamentous network containing larger spider cells and spindle shaped cells with long, brightly shining bipolar processes. This is most prominent in the left caput cavu posterior. With carmine these gliomatous areas in the gray matter stain more deeply than the surrounding gray matter.

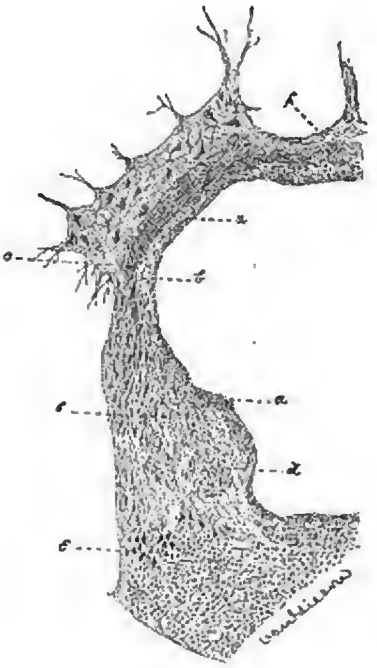


Fig. III.

In the section C VIII.–2 (Fig. 1) the cavity (6 mm. in diameter) is lined with the limiting membrane, and is bounded by the posterior commissure, the deflected column of Goll, and the left column of Burdach. The left hand portion of the wall of the cavity is in a condition of hyperplasia (Fig. 3), which makes at one point a slight nodular projection into the lumen.

On the left side of the cavity the limiting membrane merges into a zone (Fig. 3) of delicate, approximately parallel filaments containing a few larger and smaller branching neuroglia cells, and many small round and oval cells which are so intimately associated with the filaments that it is difficult to determine whether they have processes. Some of these small round cells are very likely, in such a growing mass of neuroglia, immature forms of the larger branching neuroglia cells.

The posterior portion of this filamentous zone envelops a little ball of small branching neuroglia cells whose fine processes form a loose network. This zone replaces entirely the inner four-fifths of the left column of Burdach,

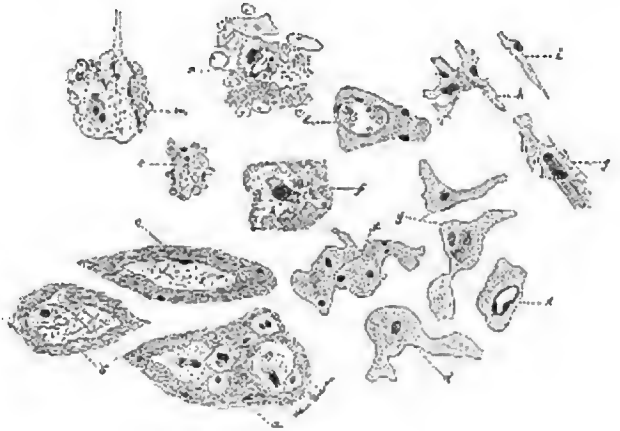


Fig. IV.

and involves to the extent shown in the drawing (Fig. 3) the gray matter where there are more neuroglia cells—most of them spindle-shaped with long bipolar processes—and fewer filaments. The right posterior horn is similarly but less extensively involved.

The hyperplastic portion of the wall also contains some small holes and spheroidal cavities, which apparently are the result of a liquefactive degeneration, and disappearance of the neuroglia cells. This seems to be so, because the various stages of the replacement of the cell-bodies by fluid are present. In some of the cells the nucleus is attached

to or surrounded by the homogeneous remains of the cell-body, and what is left of the cell-body is surrounded by fluid which stains poorly with acid fuchsin and carmine (Fig. 4 a). Other cells are converted into cavities filled with fluid (Fig. 4 b c). The gliomatous portion of the gray matter and the degenerated column of Goll contain glia cells liquefying in this way and producing little cavities. (I do not think these changes in the glia cells are produced by the action of the hardening agents, because the degenerating cells are not scattered about uniformly throughout the cord but are noticeable wherever there is much *hyperplasia* of neurogliar tissue, and in the œdematous portions of the cord described presently.)

In the apex of the left posterior horn and in the adjacent portion of the column of Burdach (Fig. 3) is a group of very much swollen and partially fluidified large glia cells, and some large irregular shaped drops of fluid. The swollen cells (Fig. 4 d) are changed into homogeneous rounded or irregular shaped neucleated lumps, occasionally having a thick, varicose, club-shaped process. Others of the degenerated cells are more or less completely changed into fluid. It is difficult to determine whether some of the large irregular drops of fluid are the result of complete liquefaction of the Deiter's cells, or whether they have perhaps leaked out of the blood-vessels. One of the smaller vessels in this region has a partial zone of fluid about its walls.

Between the sections C VIII.–2 and D III.–3 (Fig. 1) the wall of the cavity has the simpler form shown in Fig. 2. The posterior horns and the internal portion of their junction with the anterior horns, in this portion of thecord, have an increased amount of neuroglia cells and fibres. This is most marked on the left side.

In the section D III.–2 the limiting membrane is replaced by a considerably thicker zone, resembling the superficial neurogliar layer of the cord, wrapped circularly about the cavity and merging with the surrounding thickened neuroglia of the posterior columns and gray matter.

The section D III.–3 is near the upper level of a portion of the cord, in which the central portion is very œdematous.

The œdema extends nearly to the fifth dorsal segment. The wall of the cavity in this section is thickened and has about the same structure as in the preceding section. It contains liquefying glia cells and some drops of fluid. The gray matter and the adjacent portions of the lateral columns and the inner half of the posterior columns are honeycombed with drops of fluid and some granular material, which separate the elements of the gray matter and crowd apart the nerve fibres in the white matter. Besides this there are a great many swollen and liquefying glia cells (Fig. 4 y) in the œdematous areas and in the wall of the cavity. These cells are changed in the same way as already described in the section C VIII.–2. Many of them are so completely liquefied that they are not to be distinguished from the drops of fluid which have exuded from the blood-vessels. The latter are congested, and in a few places, notably in the gray matter, have several leucocytes with trifoliate nuclei in their perivascular spaces. Some of the ganglion cells are partially disintegrated.

The section D III.–4 the wall of the cavity is thickened and is composed of two zones, an inner sparingly nucleated filamentous zone wound about the cavity circularly, and an outer incompletely circular layer composed of long-armed, spindled-shaped neuroglia cells lying amongst small bundles of rather stout, glistening fibres which have a vertical direction.

In the section this outer zone has a peculiar appearance and is different from any other portion of the wall of the cavity, because the fibres are cut transversely and appear as collections of larger and smaller homogeneous, lustrous, angular discs (from 1–8 u- in diameter)separating the spindle cells. Attached to the inner wall of the cavity are some red blood-cells and a few cells looking like leucocytes. The œdema and the liquefactive condition of the glia cells is more intense and extensive than in the preceding section.

The degenerating glia cells are most prominent in the posterior horns, where in some places the swollen cells are nearly in contact (Fig. 4 x) and in other places some of the

cells seem to have coalesced, forming large, irregular shaped, faintly stained, homogeneous, nucleated lumps.

Lying against and around a very few of the smaller blood-vessels in the œdematous areas, are some homogeneous lumps of material (Fig. 4 k, l) having the same appearance as the swollen glia cells. These perivascular homogeneous lumps probably are swollen glia cells which happened to lie very close to the blood-vessels.

In the section D IV.–1 the gliomatous wall of the cavity

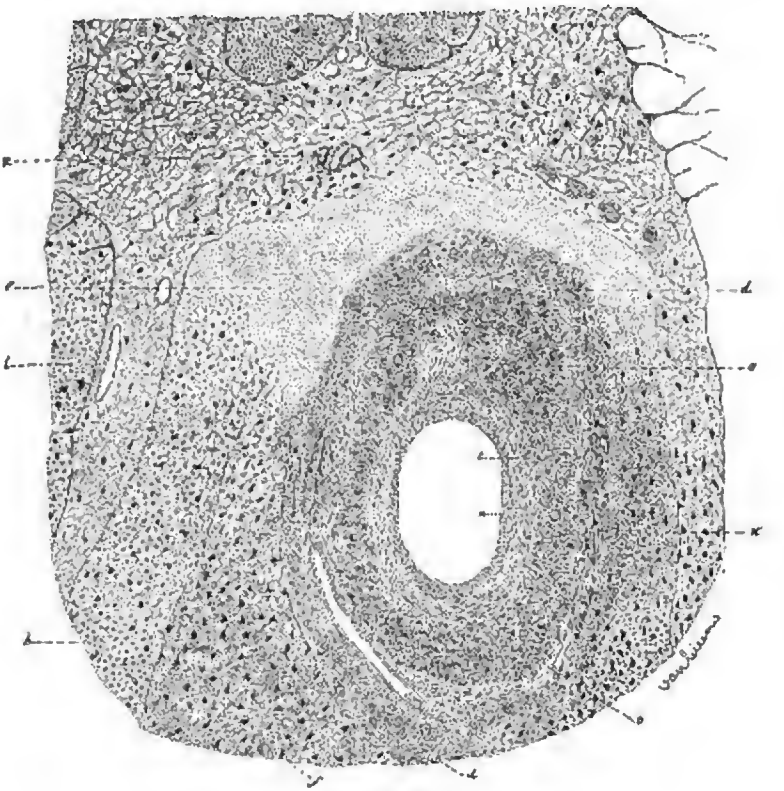


Fig. V.

containing numerous new thin-walled blood-vessels and fluidifying glia cells (Fig. 4 g, h, i)is in a more pronounced state of hyperplasia than in any other portion of the cord. The details of the structure of the wall are given in the text

of Fig. 5. About 1 mm. below this section the wall has grown into the cavity, so that the lumen is of microscopic size: The œdema and the changed glia cells are present as in the previous section. The œdema is greatest in the gray matter adjoining the commissure.

Between the outer portion of the wall and the gray commissure, and replacing a portion of the latter, is a crescentric zone consisting largely of fluid (Fig. 5), which seems to have collected from the gray matter about the dense and comparatively impervious outer layer of the cavity wall partially separating it from the surrounding tissue. This mass of fluid disappears at a slightly lower level. The wall of the cavity contains a little fluid in its meshes.

The remainder of the sections from the fourth dorsal segment do not differ materially from the previous section (D IV-1), except that the cavity gradually grows larger and its wall is much less thickened and has a simpler structure. It is composed of fine filaments with few cells, wrapped concentrically about the cavity, and the filaments merge into the surrounding thickened neuroglia. Clinging to the inner surface of the wall in the lower part of this segment (Fig. 1 D IV-4) is a mass of fluid and fibrin. The œdema ceases at the upper level of the fifth dorsal segment. The œdema seems to be recent in its origin, because it has only separated and not destroyed the elements of the cord to any great extent except the glia cells. The posterior horns and the gray commissure in the œdematous region were probably in a condition similar to that shown in Fig. 3 before the œdema took place. This is indicated by the great numbers of the changed glia cells in these œdematous portions of the gray matter (Fig. 5).

The section D V-1 shows the lesions of a chronic myelitis of the white and gray matter. The wall of the cavity, containing the changed glia cells, has about the same structure as in the lower portion of the fourth dorsal segment. A strand of fibrin stretches across the cavity.

Three mm. below this section is the upper dome-shaped extremity of the tumor (Fig. 1), which gradually growing larger and compressing the tissue of the cord into a thin

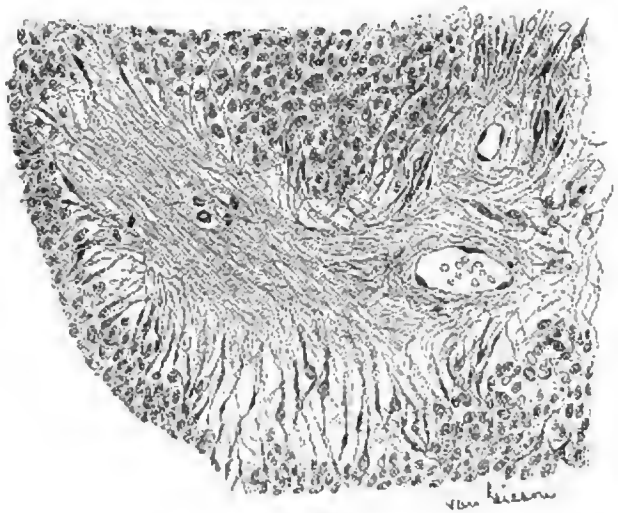


Fig. VI.

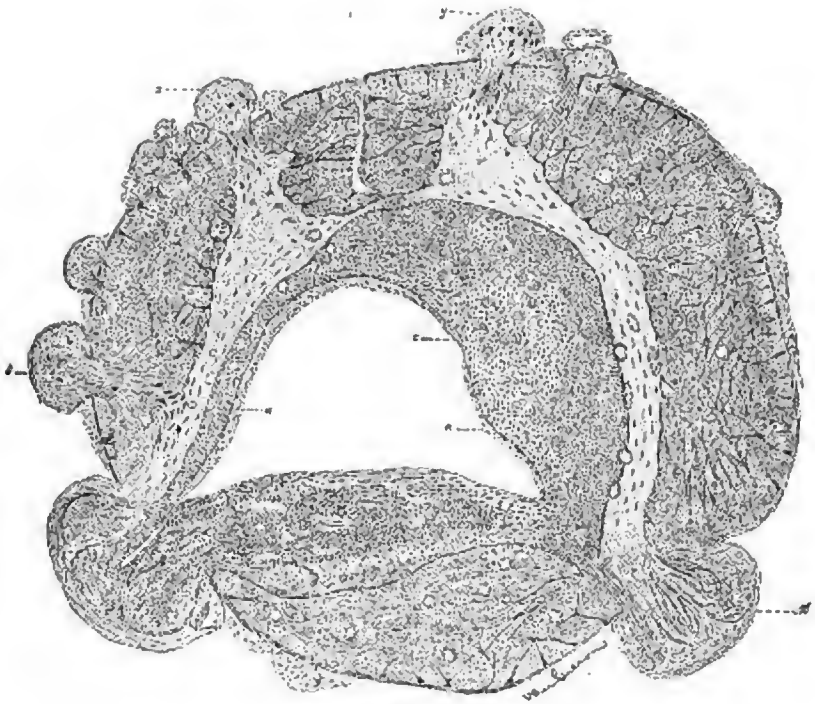


Fig. VII.

cylindrical outer shell attains its maximum diameter at D VII, where the cord measures sixteen mm., nearly twice the usual diameter of this transection.

In section, the tumor is composed of closely packed masses of spindle-shaped and oval cells associated with filaments, arranged in strands between and around numerous blood-vessels which are surrounded by a close-textured sparingly nucleated sheath of neuroglia filaments (Figs. 6, 7). When isolated, some of the tumor cells are glia cells, and other granular, rounded, or spindle-shaped non-branching cells, look like the cells of the ordinary sarcomata. The central core of the tumor is degenerated. It is composed of fluid, fibrin, hæmatogenous pigment, and, in places, of disintegrated tumor cells.

In the eighth dorsal segment the tumor does not fill up the large central portion of the cord as a solid plug, but is situated around a tubular cavity (Figs. 1, 7). Here the tumor is very distinctly separated from the cavity by a thin neurogliar membrane bearing no structural resemblance to the tumor, but identical in structure with the limiting membrane (Fig. 2). The tumor thus arranged about the cavity becomes thinner and thinner (Fig. 1), and about seven mm. below the section D VIII-2 disappears, fusing with the gliomatous wall of the cavity. The gray matter in the eighth dorsal segment, contains an increased amount of neuroglia, and there are patches of sclerosis in the white matter.

The small nodules on the surface of the cord are of three kinds (Figs. 1, 7).

In one place such a superficial nodule is produced by the stretching of the superficial neurogliar layer and a pouching out of the substance of the cord in front of a mass of tumor cells advancing toward the periphery (Fig. 1 D VIII).

Several of them are caused by direct nodular extensions of the tumor (Fig. 6).

The remainder are caused by little tumor-like hyperplasias of the cells and fibres of the neuroglia of the white matter. The latter are collected together in dense bundles

of various sizes near the periphery of the cord, and piercing the superficial neurogliar layer and pia matral covering of the cord, both the inner and outer extremities of these bundles spread out like the fibres of a broom (Fig. 7). The outer ends become tangled up in the pia mater producing little nodules, or they become fused with the neuroglia of the nerve roots which is increased at such points, and is composed of interlacing skeins of filaments with few glia cells, surrounding the nerve fibres which are diminished greatly in number. The inner ends of the bundles spread out in the gray and white matter becoming continuous with surrounding neuroglia. Between the separated ends of the fibres are generally a number of larger and smaller neuroglia cells, some of which are liquefying and have produced small cavities empty or filled with fluid (Fig. 7 b). A hypertrophy of the neuroglia accompanying the intra medullary continuations of the nerve roots forms the greater number of this third variety of the nodules.

The prominences on the roots at some little distance from the cord are due to a growth of neurogliar tissue.

In the eighth dorsal segment below the tumor the wall of the cavity containing several large blood-vessels and many liquefying glia cells, consists internally of a thin porous loose-textured inner layer and externally of a sparingly nucleated filamentous dense circular zone, which is somewhat thickened throughout the whole segment. A slender band of neuroglia stretches partly across the cavity at one place.

There are some short fibres in this portion of the wall of the cavity which have the appearance of hyaline degeneration. They are irregularly swollen, glassy, and stain brightly with dilute solutions of acid fuchsin.

The posterior horns, and the columns of Clark in places, are changed into neurogliar tissue. There is a neurogliar thickening about the posterior septum, and there are patches of sclerosis about the blood-vessels. Two columns of interstitial myelitis lying alongside of the external antero lateral margin of the anterior horns (Fig. 1) extend to the twelfth dorsal segment.

In the remainder of its extent the wall of the cavity does not differ materially from the wall in the first dorsal segment. The wall is nodularly thickened in one place (see Fig. 1 in the tracing of the cavity in D IX), and a neurogliar septum stretches across the cavity in the upper portion of the tenth dorsal segment. The right column of Clark in places is involved by the cavity wall. The cavity terminates a few mm. above D XI.

The central canal in most of its extent is normal; in places its cells are increased in number. In such places some of the cells are arranged in string-like rows, and others are grouped in a vertical tubular manner, so that in the sections they look like the lining cells in a transection of a small duct of a gland. Many of the vessels throughout the cord have a limited number of small round cells just outside of their walls. The œdema just above the tumor seems to be due to a passive effusion from the vessels. A tumor expanding the cord so extensively as this one would be liable, by interfering with the circulation, to cause an œdema in its vicinity.

Anatomical Diagnosis. — Telangiectatic glio-sarcoma occluding the middle portion of a tubular cavity in the cord, lined with neuroglia tissue which in places is in a condition of hyperplasia.

Resumé and Conclusions.—1. *There is no morphological evidence that the tumor extended beyond its present confines.* About two hundred sections of the cord above and below the tumor were examined, and in none of them is there any tissue having the structure of the tumor.

2. *There is no clinical evidence of the extension of the tumor above its upper level.* Since the symptoms did not extend above the umbilical region, the upper limit of the lesion is situated between the fourth and seventh dorsal segments.[2] This corresponds approximately to the situation of the upper level of the tumor. But the cavity is certainly large

[2] Localization of the functions of the segments of the spinal cord, in Professor Starr's paper on Syringo Myelia. American Journal of Medical Sciences, May, 1888.

enough ($\frac{3}{4}$ cm. in diameter) in the eighth cervical and first dorsal segments, had it been produced by the disintegration of a tumor, to have occasioned symptoms in the hand and forearm.

3. *The lower third of the tumor is developed outside of the inner zone of the neurogliar wall of the cavity.* In the eighth dorsal segment the tumor encircles the cavity, from which it is separated by a thin neurogliar membrane, structurally differing from the tumor, which interruptedly clothes the wall of the entire cavity internally quite extensively. This arrangement of the tumor suggests that it originated in the wall of the cavity. Possibly the upper two-thirds of the tumor grew into and occluded the lumen.

These three facts—the absence of morphological or clinical evidence of the formation of the cavity, by the tumor, and the intra-mural situation of the lower third of the tumor which is but slightly degenerated—indicate that the cavity was formed previous to the development of the tumor from the cavity wall. The cavity probably is a congenital defect; but as there are in places neurogliar hyperplasias of its wall with evidences of degeneration, the cavity has probably also been modified to some extent by the growth and disintegration of gliomatous tissue along its wall. In the places where the hyperplasia of the wall is greatest the lumen of the cavity is the smallest, and where the cavity is largest its walls are the thinnest. To what extent the cavity is congenital in its origin, and to what extent it has been enlarged by the growth and degeneration of gliomatous tissue surrounding the cavity, is difficult to determine. On account of the accommodation of the structures of the cord to the configuration of the cavity, in the developing and adult stages, no symptoms occurred until twenty months before death, when the tumor began to grow from the cavity wall. Then the regional distribution and duration of the symptoms correspond very well with the position and structural character of the tumor.

Defective closures of the developing central canal and posterior septum, with residual epiblastic tissue in the wall of the defective closure, would explain how the cavity is

formed in many cases of syringo myelia. In a section of the spinal cord of the human embryo at the sixth week, the primary central canal is relatively very large; it is diamond shaped, and extends from the anterior to the posterior surface of the cord. During the later stages of development, this primary central canal becomes divided into two portions, an anterior and a posterior portion. The anterior portion ultimately forms the permanent central canal; the sides of the posterior division coalesce and form the posterior septum. With faulty closure of either of these divisions of the primary canal there is generally some surplus epiblastic tissue lining the wall of the defect. This embryonal tissue is liable at some later time to grow, forming neurogliar hyplasias or tumors. These growths have a great tendency to disintegrate and make the cavity, which may be very limited at first, much more extensive. It is in this way very likely that many cases of syringo myelia orignate; the cavity commences in a defective closure of the divisions of the primary canal (or of the apertures through which the embryonal vessels penetrate into the cord), and acquires a greater extent by the growth and disintegration of embryonal tissue which persists along the wall of the defect. Errors in the closure of the divisions of the primary central canal as a starting-point for the cavity, explain the great frequency of the situation of the cavity, in cases of syringo-myelia, in the posterior columns or central part of the cord. In one place in this case, the cavity looks as though it was due to a defective closure of the anterior portion of the posterior septum (see Fig. 1–D VIII.–3 to D IX.–2). In these sections the anterior portion of the posterior septum is absent. Throughout the remainder of the cord the posterior septum presents no anomalies.

It is fully in accordance with Cohnheim's theory that tumors should be associated with these cavities, starting in a faulty development of the cord, and that furthermore they would be liable to arise from the wall of the cavity; for preferably in the wall of such a congenital defect we would expect the presence of residual epiblastic tissue, or of an included portion of some other inappropriate embryonic

layer, from either of which a tumor might arise. From the former condition, a neuroglioma or a neurogliar hyperplasia; in the later condition some heterologous tumor might originate.

It seems justifiable, in reviewing this case, to say that some cases of syringo-myelia are cases of congenital tubular defects in the cord, producing no symptoms until a gliomatous hyperplasia of their walls, or a tumor arising in the wall, becomes large enough to injure the cord.

The liquefactive degeneration of the glia cells in this case is interesting in connection with the breaking-down process characteristic of gliomatous tumors and certain gliomatous hyperplasias, which is so prominent a feature of these growths when they occur with syringo-myelia. The changed glia cells are so uniformly and extensively present in the hyperplastic portions of the wall as to suggest that the disintegration and disappearance of the gliomatous tissue in syringo-myelia may be, in a measure, due to such a liquefactive degeneration of the glia cells.

In the œdematous portions of the cord the changes in the glia cells are probably to a great extent secondary to the œdema; but in other portions the degeneration of the glia cells is quite independent of the œdema.

Such a condition as the œdema in this case tends to modify the configuration of the cavity. In one place the œdematous fluid has sequestrated a portion of the wall of the cavity.

The literature of syringomyelia is given in Professor Starr's paper already referred to.

EXPLANATION OF THE PLATES.

Fig. 1.—Transections from different levels of the cord and a tracing of the tumor and the cavity on the posterior surface of the cord. (The segments of the cord and their subdivisions were measured by compass, after hardening and the tracings and transections are three times the natural size—the latter were drawn with the camera lucida. The transections from D III-4 to D IV-3 have not been represented proportionately large enough). In the sections between and including D III-3 and D IV-4, the dotted areas represent the distribution of the œdema. The thicker portions of the tracing of the wall of the cavity correspond to the places where the wall is in a condition of hyperplasia.

In front of the left anterior horn in the section D VIII and attached to the posterior surface of D VII-3 are extensions of the tumor producing nodules on the surface of the cord. Corresponding to the apex of the left posterior horn in the section D VIII a superficial nodule is formed by the tumor pressing out the substance of the cord. The nodules on the anterior surface of the section D VIII-2 are hyperplasias of the neuroglia of the white matter.

Fig. 2.—A section of the limiting membrane of the cavity (from the section C VIII, Fig. 1). The upper surface bounds the lumen of the cavity, the lower mrrgin is the junction of the membrane with the surrounding white matter.

Fig. 3.—A section—a little below the section C VIII 2, Fig. 1—showing hyperplasia of the wall of the cavity. a. a., the inner or limiting membrane of the cavity; b. b., zone of filamentous neuroglia replacing the left column of Burdach, containing at d a small cluster of small spider cells; c., a zone of neuroglia cells and filaments involving the left posterior horn and inner one-half o the anterior horn; f., central canal; e., group of swollen and fluidifying glia cells.

Fig. 4.—Showing the liquefactive degeneration of the neuroglia cells from various places in the cord.

a. A group of degenerated glia cells from the hyperplastic portion (Fig. 3, b. b.) of the wall in the lower portion of the eighth cervical segment. In three of these cells a fragment of the protoplasm attached to the nucleus is surrounded by fluid. The degenerated cells are surrounded by closely woven fine neuroglia filaments; b, c, cells from the same region completely converted into fluid; d, swollen Deiter's cells from the same level of the cord (Fig. 3, e); f, g, h, i, degenerated glia cells from the hyperplastic wall of the cavity in the lower portion of the third and upper portion of the fourth dorsal segments. The remains of the cell-bodies are surrounded by fluid or lie in empty spaces; k, l, lumps of homogeneous material, surrounding two small blood-vessels, looking like the swollen Deiter's cells (from the section D IV-1); e, drops of fluid separating the nerve fibres in the œdematous white matter in D III-IV; m, n, partial liquefied Deiter's cells in the œdematous white matter in the section D IV-1. Some granular material lies about the nerve fibres near the cell n; x, y, very much swollen glia cells from the right posterior horn in the section D IV-1 (Fig. 5).

Fig. 5.—A portion of the section D IV-1 more highly magnified, showing the structure of the hyperplastic wall of the cavity, the œdematous condition of the gray and white matter and the distribution of the degenerating glia cells.

a. Inner zone of the wall, similar in structure to the limiting membrane; this inner zone is somewhat œdematous; it contains droplets of fluid and the interlacing glia fibres composing it are swollen and indistinct. b. Layer of fine filaments with small round nuclei wrapped circularly about the cavity. c. Layer of loosely packed small spider cells. d. Outermost compact layer of the wall, composed of filaments, granular matter, a few nerve fibres and very many liquefying glia cells (see Fig. 4, h, i). This outer zone at k involves the right column of Burdach where the filaments are more loosely arranged. f. Degenerated column of Goll, containing drops of fluid, degenerated Deiter's cells and cavities resulting from their liquefaction. e. Crescentric mass of fluid partially separating the wall of the cavity from the surrounding tissues. x. Cells of the central canal arranged in string-like rows. The gray matter and the white matter at h and it is honey-combed with drops of fluid which are represented by the white spaces

in the drawing. The small black spots represents the swollen and liquefied glia cells.

Fig. 6.—From a section of the tumor. Two small blood-vessels, invested by sheaths of scantily nucleated filamentous neuroglia, are surrounded by the tumor, which are so closely aggregated that the cell-bodies are not distinct.

Fig. 7.—A more highly magnified view of the section D VIII-2, showing the intra mural situation of the telangiectatic glio sarcoma, and the structure of some of the nodules on the surface of the cord.

The thin layer, a, a, lining the tumor internally is identical in structure with the limiting membrane (Fig. 2). The tumor cells are grouped about the neuroglia sheaths of the numerous blood-vessels. The area of the tumor at c is loose-textured, and contains more neuroglia cells than sarcoma cells. At the left posterior root entrance an extension of the tumor forms one of the superficial nodules. The remaining surface-nodules in this section are polypoid outgrowths of the neuroglia of the white matter. The nodule, b, contains some small cavities apparently formed by the liquefaction of the glia cells, which are represented by the black spots in the gray matter and in the nodular gliomatous hyperplasias. The larger black spots correspond to the swollen glia cells. The nodules x, y, and z are growths of the neuroglia strands accompanying the nerve roots as they enter the cord.